Bachelor Thesis

Biomedicine- Exercise physiology 180 credits

Comparison of lipid profiles based on different physical activity levels of young adults in Sweden

Exercise Biomedicine 15 credits

Halmstad 2024-06-05

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HÖGSKOLAN I HALMSTAD

Comparison of lipid profiles based on different physical activity levels of young adults in Sweden

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2024-05-30
Bachelor's Thesis in Exercise Biomedicine
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Abstract

Background: Knowledge regarding young individuals' physical activity habits and blood lipid levels can be useful to determine possible risk of developing cardiovascular disease (CVD). CVD was responsible for 32% of all global deaths in 2019 and is the most common cause of death in Sweden. This is mainly due to complications of atherosclerosis which is a multifaceted condition that progresses over a long time. CardioChek Plus is a portable instrument that can reliably measure blood lipid levels and give quick results, in combination with a global physical activity questionnaire one can easily screen individuals and provide valuable information for CVD prevention.
Aim: The purpose of this study was to investigate how different physical activity levels and time spent sedentary correlate with total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and the ratio of total cholesterol/ high-density lipoprotein cholesterol in healthy Swedish males and females aged 18-25.
Method: Fifteen healthy Swedish males and females aged 18-25 were recruited to participate in this cross sectional study. All tests were made during one occasion in the movement lab at Halmstad University. Data was collected from GPAQ to measure physical activity levels, CardioChek Plus for the blood lipids and InBody 770 to measure body composition. Statistical analyses were performed using Pearson's and Spearman's for correlation and ANOVA for linear regression analysis.
Results: 15 young adults participated in the study and consisted of 10 women and 5 men with a varying level of physical activity with a median of 3240 MET minutes/week (IQR 2060, 4540). The primary findings were a moderate correlation between time sedentary and triglyceride concentration ($r=0.623$) and a unstandardized β-value of 13.8 (CI=2.2-25.5) $p= 0.024$, which entails that TG is expected to increase by 13.8mg/dl for every unit of time spend sedentary. No relevant associations between physical activity and blood lipid concentrations were found in our tests.
Conclusion: In the studied population, time spent being sedentary was a more important factor for triglyceride levels than volume of physical activity. This suggests that one who wants to decrease their risk of developing CVD may benefit from reducing sedentary time rather than increasing physical activity. Further studies may benefit from a larger population size with a broader range of physical activity levels.

Abstrakt

Bakgrund: Kunskap om unga individers fysiska aktivitetsvanor och blodfettsnivåer kan vara användbart för att fastställa potentiell risk för att utveckla kardiovaskulära sjukdomar. Kardiovaskulära sjukdomar var ansvariga för 32% av alla globala dödsfall under 2019 och är den vanligaste dödsorsaken i Sverige. Detta beror främst på komplikationer av ateroskleros som är ett mångfacetterat tillstånd som fortskrider under lång tid. CardioChek Plus är ett portabelt instrument som på ett reliabelt sätt kan mäta blodlipidnivåer och ge snabba resultat. I kombination med ett globalt frågeformulär för fysisk aktivitet (GPAQ) kan man enkelt screena individer och ge värdefull information för att potentiellt förebygga hjärt-kärlsjukdom.

Syfte: Syftet med denna studie var att undersöka hur olika fysiska aktivitetsnivåer och tid som spenderas stillasittande korrelerar med totalt kolesterol, triglycerider, hög densitets lipoprotein kolesterol, låg densitets lipoprotein kolesterol och förhållandet mellan totalkolesterol och hög densitets lipoprotein kolesterol i friska svenska män och kvinnor i åldern 18-25.

Metod: Femton friska svenska män och kvinnor i åldrarna 18-25 rekryterades för att delta i denna tvärsnittsstudie. Alla tester gjordes under ett tillfälle i rörelselabbet vid Högskolan i Halmstad. Data samlades in från GPAQ för att mäta fysiska aktivitetsnivåer, CardioChek Plus för blodlipiderna och InBody 770 för att mäta kroppssammansättning. Statistiska analyser utfördes med Pearsons och Spearmans för korrelation och ANOVA för linjär regressionsanalys.

Resultat: 15 unga vuxna deltog i studien och bestod av 10 kvinnor och 5 män med varierande nivå av fysisk aktivitet med en median på 3240 MET minuter/vecka (IQR 2060, 4540). De primära fynden var en måttlig korrelation mellan stillasittande och koncentration triglycerider (TG) ($r=0,623$) och ett ostandardiserat β-värde på 13.8 (CI=2.2-25.5) $p=0.024$, vilket innebär att TG förväntas öka med 13.8 mg/dl för varje tidsenhet som spenderas stillasittande. Inga relevanta samband mellan fysisk aktivitet och koncentration av blodfetter hittades i våra tester.

Slutsats: I den studerade populationen var den stillasittande tiden en viktigare faktor för TG nivåer än volymen av fysisk aktivitet. Detta tyder på att individer som vill minska sin risk att utveckla kardiovaskulär sjukdom kan dra nytta av att minska stillasittande tid snarare än att öka fysisk aktivitet. Ytterligare studier kan dra nytta av en större befolkningsstorlek med ett bredare spektrum av fysiska aktivitetsnivåer.

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Abbreviation list

Apo; Apolipoproteins

ABCA1; ATP-binding cassette transporter

BF%; Body fat percent

BIA; Bioelectrical impedance analysis

BMI; Body mass index

CT; Computerized tomography

CVD; Cardiovascular disease

DXA; Dual-energy x-ray absorptiometry

ECD; Endothelial cell dysfunction

eNOS; Endothelial-derived NOS

FA; Fatty acids

FFM; Fat-free mass

FH; Familial hypercholesterolemia

FM; Fat mass

GPAQ; Global Physical Activity Questionnaire

HDL-C; High-density lipoprotein cholesterol

IDL-C; Intermediate-density lipoprotein cholesterol

IL-1; Interleukin 1

LDL-C; Low-density lipoprotein cholesterol

LDLR; Low-density lipoprotein cholesterol receptor

MCP-1; Monocyte chemotactic protein-1

MET; Metabolic equivalent of task

MRI; Magnetic resonance imaging

MMP; matrix metalloproteinases

mRNA; Messenger ribonucleic acid

NCEP ATP III; National cholesterol education program adult treatment panel III

NOS; Nitric oxide synthase

PA; Physical activity

PCSK9; Proprotein convertase subtilisin/kexin type 9

POC; Point-of-care

TC; Total cholesterol

TG; Triglycerides

TNF; Tumor necrosis factor

VLDL-C; Very low-density lipoprotein cholesterol

WHO; World health organization

1. Background

1.1 Introduction

Globally, there is a rise in cardiovascular diseases (CVD) and was in 2019 responsible for 32% of all global deaths (WHO, 2021). In Europe it is estimated that 30% of the people under the age of 65 die in some form of CVD, where atherosclerosis is the leading cause (Kopčeková et al., 2020). In Sweden currently, the most common cause of death is cardiovascular disease (Socialstyrelsen, 2022). Why individuals develop atherosclerosis is still not fully clear, but there are some risk factors and habits that have been identified to raise the chance of developing it. The main risk factors that contribute regards: high-serum total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and low levels of high-density lipoprotein cholesterol (HDL-C), but also diabetes, obesity, sedentary lifestyle, hypertension and smoking (Kopčeková et al., 2020).

1.2 Cholesterol

Fatty acids (FA) originate mainly from dietary triglycerides (TG) or endogenous sources such as adipose tissues and de novo-synthesis via carbohydrate metabolism. TG is first emulsified in the intestinal lumen and gets hydrolyzed by lipase produced by the pancreas, yielding the lipid molecules sn-2-monoacylglycerols and free FA (Figure 1). Lipid molecules are then resynthesized into TG via esterification of sn-2-monoacylglycerols and free FA which are transported via chylomicron molecules (Alves-Bezerra et al., 2017). An important aspect of lipid molecules are apolipoproteins (Apo) which serve as ligands for lipoprotein receptors, activate and inhibit lipoprotein metabolism, guide lipoprotein formation and are a part of the core structure of the lipid molecules (Feingold 2024).

Chylomicrons are large particles, rich in TGs synthesized in the intestine, which have a function of transporting cholesterol and TGs from the diet to the liver and peripheral tissues. The chylomicrons consist of several different apolipoproteins, where Apo B-48 is the core structural protein. The size of the chylomicrons is dependent on the meal consumed, a meal rich in fat leads to enlarged chylomicron particles, due to the increase in TGs. When the enzyme lipoprotein lipase in the peripheral tissues unload the TGs from the chylomicrons it results in smaller particles called chylomicron remnants, which are cholesterol rich particles that are classified as pro-atherogenic (Feingold, 2024). The liver is the main organ involved in the homeostasis of lipids via strict regulation pathways. In the liver there are hepatocyte cells which are the most prominent cell in the

liver. Hepatocytes control liver metabolic functions revolving carbohydrate, lipid and proteins. In hepatocytes the TG from chylomicrons gets hydrolyzed into FA which in turn are activated to form acetyl-CoA molecules which are involved in the synthesis of cholesterol which is a type of sterol (Alves-Bezerra et al., 2017).

Sterols are fat soluble alcohols, where cholesterol is the most common of them. It is built up with a ring structure of 17 carbon atoms where methyl groups are bound to carbon 10 and 13 and a hydrocarbon chain bound to carbon 17. Cholesterol has important functions for the human body. It is an important precursor for synthesis of steroid hormones (such as cortisol and sex hormones), vitamin D and bile acids. It also has an important role in the cell membranes and in lipoproteins. In cell membranes it influences the permeability of which substances that can pass, in cooperation with proteins and phospholipids. The metabolism of cholesterol is controlled efficiently in healthy individuals, with the mechanism of an inhibition of the endogen synthesis if we consume an excess of cholesterol, whereas lack of cholesterol activates it. However, in individuals with different metabolic disturbances this control mechanism could get affected and cause problems (Abrahamsson et al., 2013).

1.3 Lipoprotein

Very low-density lipoprotein cholesterol (VLDL-C) are TG rich particles produced in the liver (figure 2). VLDL-C particles are smaller than chylomicrons, but the mechanism of its size works in a similar way, it varies depending on the presence of TGs in the particle (figure 3). This means, when the liver increases the synthesis of TGs, the VLDL-C particles that's being secreted are bigger. When VLDL-C reaches muscle- and adipose tissue and releases the TGs it changes formation into a cholesterol-rich particle called intermediate-density lipoprotein cholesterol (IDL-C). From these pro-atherogenic particles and VLDL-C, a particle called LDL-C is derived, which carries the larger part of the cholesterol in the body's circulation. The main structural apolipoprotein in LDL-C, VLDL-C and IDL-C is Apo B-100 which serves as a ligand for the LDL-C receptor (LDLR). LDLR is important regarding clearance of lipoproteins from the circulation. High levels of Apo B-100 indicate a greater risk of developing atherosclerosis. Mutations in this apolipoprotein is associated with a genetic disease called familial hypercholesterolemia (FH), this due to less affinity for the LDLR. The size and density of LDL-C particles can vary, where small dense LDL-C particles seem to be more pro-atherogenic than larger particles. Hypertriglyceridemia, obesity, type 2 diabetes, low HDL-C levels and inflammatory states are all associated with an abundance of small dense LDL-C. There are a few reasons why they are considered more pro-atherogenic: these particles have less affinity for the LDLR than larger LDL-C particles, which leads

to a prolonged retention time in the circulation. Also, they can pass through the walls of arteries more easily and stick to the arterial wall by binding to intra-arterial proteoglycans. At last, these particles are more easily oxidized, which could attract macrophages and result in an increased uptake (Feingold, 2024).

In most tissues of the body, but mainly in the liver, LDLRs are present and mediate uptake of the pro-atherogenic particles chylomicrons, IDL-C and LDL-C through endocytosis into the cell. Once in there, lysosomes break them down and cholesterol is released. When cholesterol gets delivered to the cell, the activity of HMG CoA reductase decreases, as well as other enzymes required for biosynthesis of cholesterol and the expression of LDLRs. The amount of cholesterol present in the cell regulates the quantity of LDLRs present in the liver. Therefore by determining the number of hepatic receptors, the plasma levels of LDL-C could be estimated. High concentration of receptors is associated with low levels of LDL-C in the circulation, while low quantity of receptors in the liver is associated with high levels of LDL-C (Feingold, 2024).

Besides the earlier mentioned pro-atherogenic particles, there are proteins called HDL-C which are considered anti-atherogenic. They have a key role in the mechanism of reverse cholesterol transport from peripheral tissues to the liver. HDL-C particles contain a lot of cholesterol and phospholipids and the core structural apolipoprotein is Apo A-I, which is associated with less risk of developing atherosclerosis. The inhibiting effect on atherosclerosis could be due to the particles anti-oxidative, anti-inflammatory, anti-thrombotic and anti-apoptotic properties (Feingold, 2024). Therefore a high level of HDL-C in the blood is something to strive for, in a combination with low LDL-C (Linton et al., 2019).

Healthy levels of LDL-C for both men and women aged 20 or older is less than 100 mg/dL, and HDL-C-levels should be >40 mg/dL for men and >50 mg/dL for women (MedlinePlus, 2020). Individuals with elevated TC levels (>200 mg/dL) have been seen to have about double the risk of encountering coronary heart disease, peripheral artery disease and cerebrovascular disease compared to individuals with healthy TC levels. In Europe this applies to 54% of adults aged ≥ 25 years who have TC levels above the recommended levels (Mann et al., 2014). Since there could be individual differences it is relevant to observe the ratio between TC/ HDL-C. According to Bailey (2022) a ratio below 5:1 is desirable, but below 3.5:1 is optimal.

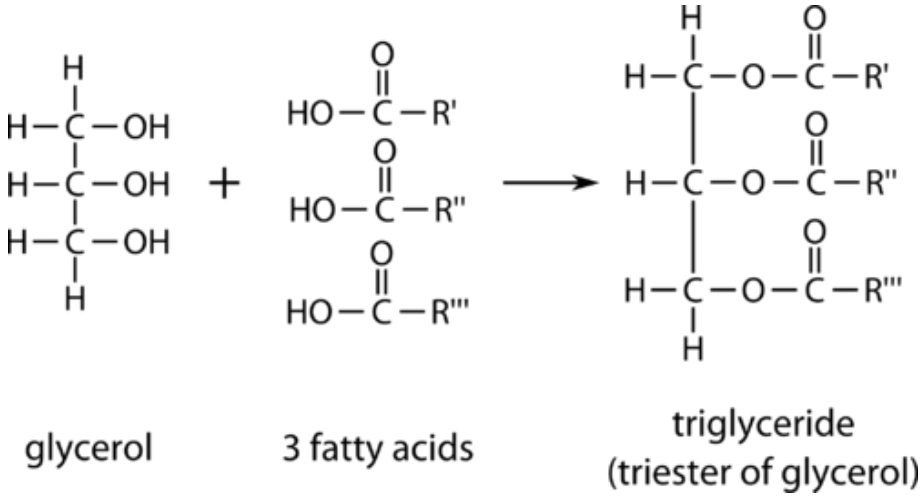

Figure 1. Structure of triglycerides (Chemistry LibreTexts, 2024)

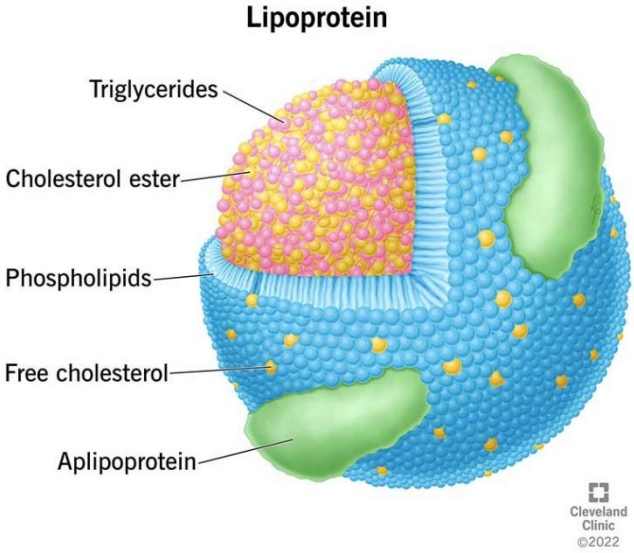

Figure 2. General structure of a lipoprotein (Cleveland Clinic, 2022)

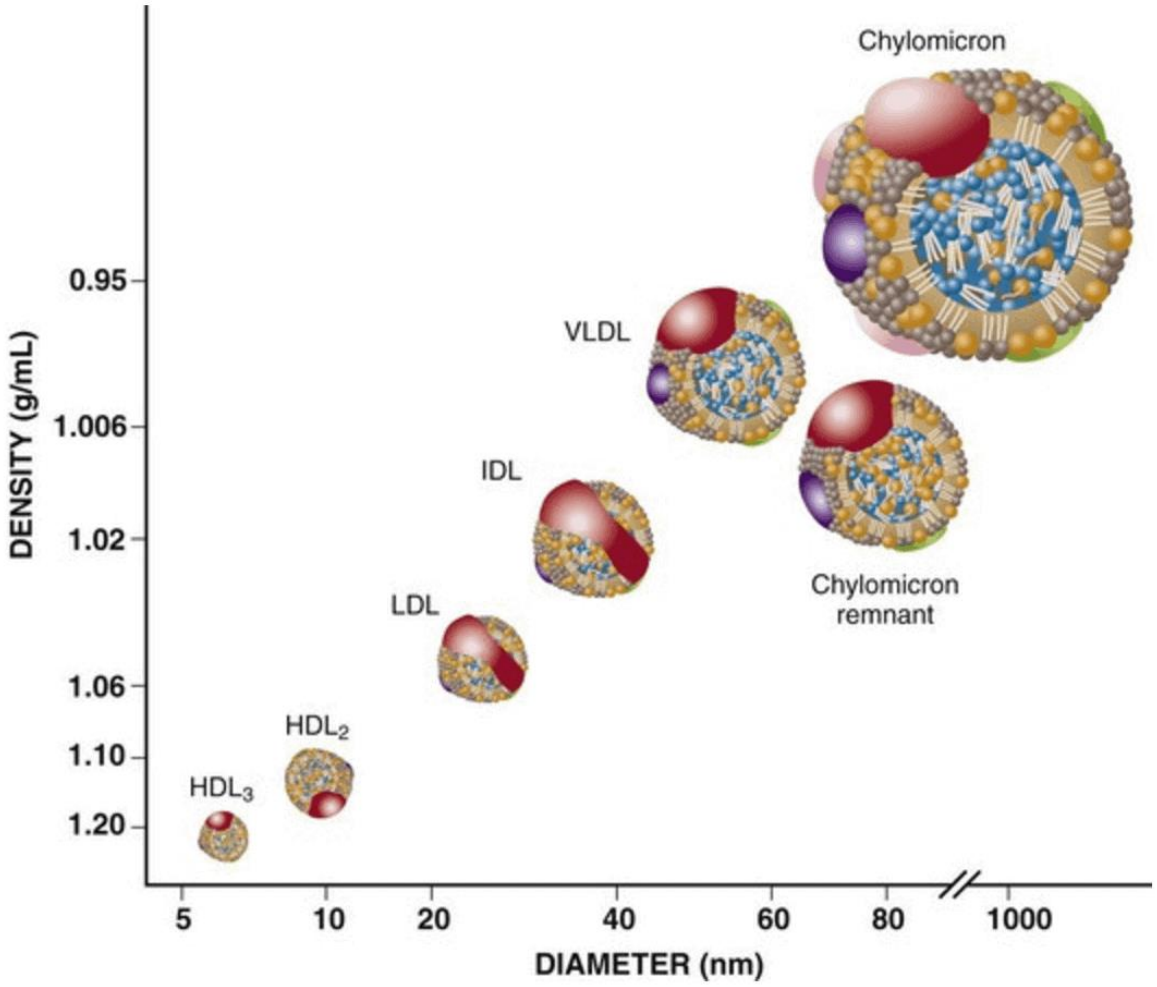

Figure 3. Density and diameter of lipoproteins (Sigurdsson, 2017)

1.4 Pathology of cholesterol related cardiovascular disease

1.4.1 Metabolic syndrome

Metabolic syndrome is a medical term that refers to a group of risk factors that contribute to an increased risk of CVDs. The risk factors include obesity, hyperglycemia/ insulin resistance, atherogenic dyslipidemia and hypertension. These conditions share underlying mediators, mechanisms and pathways and are interrelated (Huang, 2009). The pathophysiologic state originates mainly from an imbalance in calorie intake and energy expenditure, but is also influenced by other factors, such as sedentary lifestyle, genetics and epigenetics (Saklayen, 2018). The most widely used definition of metabolic syndrome is from the national cholesterol education program adult treatment panel III (NCEP ATP III), managed by the National Institutes of Health. This definition means that metabolic syndrome is present if three or more of the following five criteria are met: waist circumference >40 inches (101.6 cm) in men and >35 inches (88.9 cm) in women, fasting glucose ≥100 mg/dL, TG ≥150 mg/dl, HDL-C <40 mg/dL in men and <50 mg/dL in women and blood pressure ≥130/85 mmHg (Huang, 2009).

Abdominal obesity as previously mentioned is associated with metabolic syndrome, CVD and is also a risk factor for all-cause mortality. The golden standard method measuring abdominal adipose tissue is by computerized tomography (CT) and magnetic resonance imaging (MRI). Another method more widely used in large-scale studies is to measure waist circumference of the individual. The strengths of this method is its low cost, its simplicity to use and is applicable to all body sizes (Fang et al., 2018). Values of a waist circumference >94cm (men) and >80cm (women) indicate an increased risk of metabolic complications (WHO, 2008).

1.4.2 Hypertension

One of the main contributors to premature morbidity and mortality in the United States is hypertension (Elliott, 2007). Hypertension is defined as continual high blood pressure ≥140/90 mmHg according to WHO (2023). Factors that higher the risk of having elevated blood pressure include genetics, older age, being overweight or obese, high-salt diet, drinking lots of alcohol and not being physically active. Some of these risk factors are modifiable, such as the diet, being overweight, sedentary lifestyle and the consumption of alcohol and tobacco. However, there are also risk factors that are non-modifiable including age, genetics and concurrent diseases like diabetes or diseases related to the kidneys. Symptoms of hypertension could be severe headaches, chest pain and dizziness for instance, although the majority of people don't feel any symptoms at all. Complications of hypertension that’s uncontrolled or untreated including heart related diseases, stroke and kidney disease (WHO, 2023).

1.4.3 Endothelial dysfunction

The vascular endothelium, which consists of an endothelial lining of blood vessels functions as a selective permeable interface, regulating transport of macromolecules and fluids into the underlying tissues. Endothelial cell dysfunction (ECD) is characterized by a localized loss in the permeability function of the endothelium lining, allowing LDL-C to enter the subendothelial space, where they might be oxidized and become cytotoxic, proinflammatory and proatherogenic (Falk, 2006).

ECD is suggested to be causal with several factors such as hypercholesterolemia, diabetes mellitus (diabetes type-2), metabolic syndrome, hypertension, sex hormonal imbalance, aging, oxidative stress, proinflammatory cytokines such as interleukin 1 (IL-1) and tumor necrosis factor (TNF), smoking, air pollution, bacterial infection, viruses and disturbed blood flow (Gimbrone et al., 2016).

Observations show that nitric oxide synthase (NOS) may be atheroprotective or atherogenic depending on its production source. Endothelial-derived NOS (eNOS) is produced by the endothelial cells functions as inhibitor of activation adhesion and aggregation in leukocytes, causes vasorelaxation in the vascular smooth muscle tissue and reacts with hemoglobin to enhance oxygen delivery to tissues. Observations from animal and human studies show that deficiency of eNOS production impairs flow mediated vasodilation and may proceed the atherosclerotic plaque formations. NOS produced in a higher capacity by macrophages has more potent oxidative properties and may be atherogenic if combined with LDL-C in the arterial intima (Gimbrone et al., 2016).

1.4.4 Atherosclerosis

Atherosclerosis is primarily an inflammatory disease characterized by a plaque formation consisting of many different components in the intima layer of the artery blood vessel, obstructing blood flow more and more during its long progression. The disease may be asymptomatic for several years before having a clinical effect on the individual as the condition progresses (Mushenkova et al., 2020). According to Mushenkova et al (2020) the primary risk factor for atherosclerosis development is elevated TC levels in the plasma, however many more factors impact the development of this multifocal disease. The pathogenesis of the disease starts primarily in areas with ECD, where LDL-C may pass through the endothelial due to the increased permeability and enters the intima, where it gets oxidized. The oxidized LDL-C particle is unable to exit the intima which results in a chemotaxic, cytotoxic and proinflammatory response. The endothelial cells get activated by the inflammatory stimuli caused by oxidized LDL-C, and up-regulates vascular adhesion molecule-1 expression which recruits monocytes and T lymphocytes that pass through into the intima.

In the intima the migrated monocytes differentiate into macrophages which via scavenger receptors internalizes oxidized LDL-C, the macrophages eventually become foam cells due to their large amounts of cholesteryl esters giving them a foam-like appearance. Intracellular cholesterol accumulation in the macrophages and foam cells will not downregulate the scavenger receptors function, resulting in continued internalization of cholesterol eventually leading to the apoptosis or necrosis of the cells. The cellular content then makes up the necrotic core of the plaque, consisting of the cellular debris and lipids (Falk, 2006).

The further the plaque expands, the more it puts pressure on the endothelial layer and obstructs blood flow of the lumen. Macrophages and T-cells also release cytokine-

signaling complexes, mainly monocyte chemotactic protein-1 (MCP-1). MCP-1 recruits dendritic cells, mast cells, B-cells and natural killer T-cells to the area. These inflammatory cells also get activated and release interferon gamma and TNF further driving the inflammation (Vacek et al., 2015).

Macrophages and endothelial cells also contribute more atherogenic and atheroprotective effects by excreting proteolytic enzymes such as matrix metalloproteinases (MMP) which are zinc-containing endopeptidases. MMPs are involved in vascular remodeling via degradation of the extracellular matrix separating the intima and the media layer of the blood vessel, allowing smooth muscle cells to migrate into the intima layer and proliferate (Vacek et al., 2015). If the disease persists and progresses, the smooth muscle cells will mediate proliferation of fibrous tissue consisting of collagen and fibroblasts making up a fibrous cap surrounding the lipid rich plaque. This fibrous cap has protective properties since the plaque has a high thrombogenicity. There are two main factors that determine the severity of the atherosclerosis plaque, the size of the necrotic core and the thickness of the fibrous cap. The relationship between them determines the chances of a rapture in the fibrous cap (Alonso-Herranz et al., 2023).

If the fibrous cap ruptures, it may become fatal, due to the collagens and necrotic thrombogenic material spilling out into the lumen. The coagulation system will respond, activating thrombocytes which will start clotting at the point of rupture which may accumulate into a thrombus (Alonso-Herranz et al., 2023). Thrombuses can enlarge until the lumen is partially blocked or completely blocked, restricting blood flow. The severity of this thrombosis depends on the location of the blockage, with myocardial infarction, cerebrovascular accident or stroke having the highest mortality (Ashorobi et al., 2023).

1.5 Factors that influence cholesterol levels

1.5.1 Genetics

FH is a common metabolic disorder which is characterized by elevated LDL-C levels and an increased risk of premature diseases related to the cardiovascular system in both men and women. The prevalence of the disorder according to recent genetic studies is about 1 in 200-250. FH is due to mutations in genes that code for the LDLR, such as apolipoprotein B (ApoB) which causes a defect binding region, which results in a decreased capacity to clear LDL-C from the circulation. Individuals who suffer from this genetic disease have a significantly higher risk of developing atherosclerotic complications, although early diagnosis and treatment reduces the risk (Bouhairie &

Goldberg, 2015).

Many steps are required to synthesize new mature HDL-C particles. One important step in the buildup is when the main apolipoprotein in the particle is being synthesized, Apo A-I which works as a structural protein. Cholesterol and phospholipids from the hepatocytes and enterocytes are obtained to the Apo A-I after it has been secreted, which is facilitated by ATP-binding cassette transporter (ABCA1). In Tangiers disease, the patients have lost some function in this protein due to mutations which leads to a failure of lipidate the newly synthesized Apo A-I. This results in an accelerated catabolism of Apo A-I and very low levels of HDL-C in these individuals (Feingold, 2024).

1.5.2 Diet

Lipoprotein levels are influenced by several different dietary constituents, which makes the diet important in the sense of cardiovascular disease development. Saturated FA, trans FA and cholesterol derived from a dietary source increases LDL-C. On the other hand, fibers, phytosterols, monounsaturated FA and polyunsaturated FA decrease levels of LDL-C. The effect of dietary cholesterol on LDL-C levels is modest, and varies between individuals with approximately 15-25% (Feingold, 2021).

Major sources of monounsaturated FA in the diet comes for instance from olive oil, sesame oil, avocados, peanut butter and several seeds and nuts, while sources of polyunsaturated FA are partly found in soybean oil, sunflower oil, walnuts, tofu and soybeans. The mechanism behind the decrease in LDL-C from the unsaturated FA is because it increases hepatic LDLR activity and abundance of protein and messenger ribonucleic acid (mRNA), which in turn increase clearance from circulating LDL-C. When free cholesterol from the liver decreases it results in an up-regulation of LDLR expression, leading to lowered LDL-C levels. The membrane fluidity becomes greater with an impact from polyunsaturated FA, leading to an increased ability for the LDLR to bind LDL-C (Feingold, 2021).

The trans FA main dietary sources are meat and dairy products. There has been nearly a linear relationship between LDL-C levels and intake of trans FA. It also reduces levels of HDL-C (Feingold, 2021).

Dietary cholesterol is found in food such as beef, pork, cheese and egg yolks for instance. Fibers are carbohydrates that are non-digestible and are mainly found in vegetables, fruits, whole grains, nuts and legumes. Natural occurring constituents of plants are the phytosterols and are consumed in the diet from vegetable oils, nuts,

cereals, fruit and vegetables (Feingold, 2021).

Comparing different diets, data shows that individuals consuming vegetarian diets tend to have better cardiovascular markers compared to omnivores, with a reduced risk of morbidity and mortality from ischemic heart disease, as well as other diseases such as different types of cancers, type 2 diabetes and metabolic syndrome, especially in vegans. Dietary interventions where the participants consume vegetarian and vegan diets demonstrate improvements in lipid profiles, such as a lowered TC, TG and LDL-C (Lynch et al., 2018).

1.5.3 Body composition

Body composition estimates (body fat percent (BF%), fat mass (FM) and fat-free mass (FFM)) is used in both professional and medical settings when evaluating conditions such as obesity, hydrations status and sarcopenia for instance (McLester et al., 2020), as well as the effect of various interventions related to diet, physical activity and supplementation (Antonio et al., 2019). Reliable values of this, makes it possible for patients and clients to provide proper assessments of their current health status or even risks of their health (McLester et al., 2020). Overweight and obesity are risk factors in individuals associated with an earlier development of CVD (Singh et al., 2021). A normal BF% for men is ranging between 8-19% for individuals between 20-39 years, and higher in women, between 21-33%. Below this range is classed as underweight, and higher is identified as overweight (Gallagher et al., 2000).

A method used for assessing body composition is through bioelectrical impedance analysis (BIA) which compared to other methods is relatively quick, simple and doesn't expose the individual to radiation. One type of BIA device is a multi-frequency bioelectrical impedance instrument called InBody 770. Measurements from InBody 770 include values of an individual's body weight, FFM, FM and BF%. Comparing the instrument to dual-energy x-ray absorptiometry (DXA) which often is used as a criterion method, InBody 770 overestimates FFM and underestimates FM and BF%. However, there were no statistical significant differences between the two instruments when determining changes in FM, FFM and BF% over a period of four weeks and may be a feasible alternative to the DXA (Antonio et al., 2019).

1.5.4 Medicines

Levels of LDL-C can be lowered by drugs such as statins, ezetimibe, proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors, bempedoic acid and bile acid sequestrants. The mechanism behind this is by increasing the uptake of LDL-C through

an increased synthesis of hepatic LDLR (Feingold, 2021). Statins inhibit the enzyme HMG-CoA reductase which restricts the biosynthesis of cholesterol and decreases the cholesterol concentrations in the liver. This results in increased expression of LDLRs in the membrane of liver cells, which reduces the levels of LDL-C particles in the circulation (Sirtori, 2014). Statins are recommended to be the initial drug for individuals with FH (Bouhairie & Goldberg, 2015).

As previously mentioned the quantity of LDLRs regulate the uptake of LDL-C to the hepatocytes. A key regulator of how many receptors present in the liver is the protein PCSK9. The mechanism behind this is that it binds to the LDLR and forms a complex, resulting in endocytosis and the receptor is degraded in lysosomes of the hepatocytes. Since PCSK9 reduces the amount of LDLRs, it results in increased levels of LDL-C. The relatively new protein discovered in 2003 led to a new developed drug called PCSK9 inhibitors. This drug inhibits the synthesis of PCSK9 which promotes the presence of LDLRs and clearance of LDL-C from the circulation (Grześk et al., 2022).

1.5.5 Physical activity

According to many long-term studies, increased physical activity is highly associated with all-cause mortality reduction and may also increase one's life expectancy which is linked with CVD development. Individual cardiorespiratory fitness levels have been found to be inversely related to death rates even in the presence of predictors such as hyperlipidemia, smoking and hypertension. Physical activity has shown to impact many important biomarkers for cardiovascular health (Nystoriak et al., 2018).

Physical activity in the form of resistance training or a combination of aerobic and resistance training has shown to decrease TC and LDL-C (Mann et al., 2014). Aerobic exercise which involves large muscle use during long durations has shown to influence blood lipid metabolism by increasing lipoprotein lipase concentration and activity in skeletal muscles which in turn increases HDL-C levels. Aerobic exercise has also had an accelerating effect on lipid transfer and lowers TC levels (Franczyk et al., 2023). Physical activity also appears to affect the levels of TG response favorably, however the effect is greater in individuals with high baseline TG than those with healthy levels (Madan & Sawhney, 2024).

Cardiac stroke volume and heart rate increase cardiac output during exercise, which in turn elevate the arterial blood pressure, while increased blood pressure is a risk factor for cardiovascular disease, long-term exercise promotes an overall reduction in resting blood pressure. According to Fagard (2001) observations of the results of 44 randomized

controlled trial studies, performing moderate to intense exercise 3-5 times a week for 4 weeks lowered the average blood pressure by 3.4/2.4 mmHg on average in the studied population. Currently, WHO recommends a minimum of 600 metabolic equivalent of task (MET) minutes of total activity per week to achieve health benefits. This activity level represents approximately 150 minutes of brisk walking or 75 minutes of running in a week (Kyu et al., 2016). According to Kyu et al (2016) the physical activity level should be several times higher than the minimum recommendations from WHO, to decrease the risk of getting diseases such as breast cancer, colon cancer, diabetes, ischemic heart disease and ischemic stroke. They suggest that most health gains occur when individuals reach a physical activity level at 3000-4000 MET minutes/ week.

Being physically active as a child and adolescent in the age of 6-17 has many health benefits, such as positive effects on weight status, improved muscular and cardiorespiratory fitness, bone and cardiometabolic health. The evidence existing right now suggests that those health benefits we get from physical activity in adolescence will carry on into adulthood. According to recommendations from WHO and 2018 US guidelines, children and adolescents are recommended to do at least 60 min of moderate-to-vigorous physical activity daily to achieve these benefits (Piercy et al., 2018). According to the Public Health Agency of Sweden (2024) 26.4% of Swedish men and women aged 16-29 did not reach the WHO's goal of 600 MET minutes/week and 56.5% did not exceed 1200 MET minutes/week.

In a study by Celis-Morales et al (2012) they studied the associations between CVD markers and physical activity levels in 317 adults using physical activity data. The results showed that the time spent sedentary was positively associated with increased levels of TC, TG, LDL-C and negatively associated with HDL-C levels even when they adjusted for time spent performing moderate-to-vigorous physical activity. This suggests that reducing time sedentary may be more important than time spent physically active for reducing TC, TG and LDL-C and increasing HDL-C.

Intervention studies and projects that regard physical activity (PA) levels of individuals need valid and reliable tools. Pedometers and accelerometers are technological tools that measure PA objectively and have shown to have a high accuracy. Methods that are more low cost and have the advantage to reach out to a larger population, are self-reported PA questionnaires. However, there are limitations with some of the questionnaires. One main issue is that the questions are mainly focused on the individual's PA in their leisure time, and doesn't take into account the PA performed to, during or from work and people tend to overestimate their physical activity level and underestimate their sedentary

behavior. The Global Physical Activity Questionnaire (GPAQ) is a self-reported questionnaire consisting of 16 questions which revolves around three domains: Occupational, transport-related and leisure-time PA combined with the individual's sedentary behavior (Keating et al., 2019). GPAQ was developed 2002 by WHO and has by undergoing a research programme been shown to be valid and reliable. The questions in the questionnaire have been developed and customized to suit over 100 different countries, and take consideration to cultural differences (WHO, 2023).

1.6 Methods to measure lipids

β-quantification is considered the gold standard for measuring serum LDL-C in patients and is done by separating the lipoproteins by utilizing poly-anions and ultracentrifugation. However, this method is expensive and has a high resource demand. In routine practice the most common method to measure LDL-C is the Friedewald formula. Friedewald's formula has a high correlation with β-quantification but is limited since the subject needs to be fasting since it assumes that VLDL is constant (Krishnaveni et al., 2015).

For individuals with normal fasting triglycerides (<150 mg/dl) that consumed a meal consisting of 20g fat, the postprandial triglycerides increased by an average of 106 mg/dl approximately 2-3 hours after the first meal of the day and fasting triglycerides should be measured at least 8 hours after a meal (Keirns et al., 2021). However, in a study by Nordestgaard et al (2016) they compared fasting lipid levels with non-fasting lipid levels. The observed results showed that TG, and LDL-C was increased in the non-fasting population +26 mg/dl and +8 mg/dl respectively. However, the authors proclaim that the increases are clinically insignificant and are comparable to non-fasting values when used as predictors for CVD development, even though the use of Friedewald's formula becomes less valid for calculating LDL-C. Fasting lipid levels may be more necessary in patients that exceed the recommended levels which is not relevant in the scope of this study.

Roche Cobas analyser is the standard laboratory instrument within healthcare for measuring TC, HDL-C and TG, it then calculates LDL-C with Friedewald's formula. Limitations of the Cobas analyser is that it is stationary and expensive. A more suitable instrument for quicker and more mobile testing is the point-of-care (POC) testing device Cardiochek Plus (Figure 4) that is able to be transported and gives fast results. The industry standard accuracy of measuring TC, HDL-C and TG is ±10%, ±12% and ±15% respectively, when Cardiochek Plus was compared to Roche Cobas analyser it fell within acceptable range of accuracy with -7.8%, -6.2% and 5.1% accordingly, which

makes it suitable for POC tests. Cardiochek also calculates LDL-C with Friedewald's formula (Bastianelli et al., 2017).

Figure 4. CardioChek Plus (Pts diagnostics, 2024)

Prior studies have shown that the CardioChek Plus does not differ significantly from the laboratory method using Cobas when measuring lipid profiles. However, the CardioChek plus systematically underestimates HDL-C levels (Bastianelli et al., 2016; Giampietro et al., 2007). Strengths of the CardioChek plus compared to the laboratory counterpart Cobas is its portability and quick analyses while still having high reliability (Bastianelli et al., 2016).

1.7 Rationale

TC, TG, LDL-C, HDL-C and the TC/HDL-C ratio are important biomarkers for development of CVDs, especially in atherosclerosis. Since blood lipids are dependent on the activity levels of the individual it is of interest to examine to what degree METS and sedentary behavior impact these values for the younger population. This is especially relevant since many Swedish men and women aged 16-29 do not reach the already low recommendations of physical activity. Using a portable testing device for blood lipids may also open up the possibility for screening options that are more accessible for non-

clinical settings. To our knowledge no prior research has looked at the relationship between physical activity/ inactivity and the lipid profile of young adults in Sweden.

1.8 Aim

The aim of this study was to investigate how different physical activity levels correlate with total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and the ratio of total cholesterol/ high-density lipoprotein cholesterol in healthy Swedish males and females aged 18-25.

1.9 Research question

To what degree do physical activity levels and sedentary time correlate with concentrations of total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and total cholesterol/ high-density lipoprotein cholesterol ratio in healthy Swedish males and females aged 18-25?

2. Method

2.1 Subjects and study design

This cross-sectional study tested the lipid profile of Swedish males and females aged 18-25 from Halmstad university, 10 women and 5 men (n=15). Subjects were recruited via mail, text and/or verbally and were mainly found via personal connections or via Högskolan i Halmstad. Individuals who smoke, were pregnant, had metal implanted, had a CVD related diagnosis, familial hypercholesterolemia or took medicine impacting cholesterol regulation, were excluded from participating in this study. Prior to the tests, subjects were instructed to fast for at least 2 hours and not exercise the night before. All tests were made during one occasion in the movement laboratory between 8:00-10:00 at Halmstad University and the total test time was approximately 20 minutes.

2.2 Testing procedure & data collection

2.2.1 GPAQ

Initially the participants were asked to rest seated for 10 minutes, while signing the informed consent that they had read before arrival of the testing day (see Appendix A). They were then instructed to fill in the GPAQ to assess their subjective physical activity levels in MET minutes/ week (see Appendix B). Subjects were excluded if they

provided values that were inconsistent, implausible or performed >16 hours of vigorous work, moderate work, transport, vigorous recreation, or moderate recreation activity per day (WHO, 2024).

To calculate the MET minutes/ week of the three domains from the GPAQ, moderate intensity of physical activity corresponds to a MET value of 4, while vigorous intensity corresponds to a value of 8. Cycling and walking was also classified as a MET value of 4. The durations of the different types of physical activity were multiplied by these MET values and then added up together to get the participants total MET minutes/ week (WHO, 2024).

2.2.2 CardioChek Plus

First the participant's' fingertip were wiped with alcohol and dried with paper before puncturing the middle-/ ring finger with a lancet depending on what the participant preferred. The first drop of blood was wiped off, then capillary blood was absorbed by a micropipette and added to the measuring stick of the CardioChek Plus. The instrument provided measurements of the subjects TC (mg/dl), TG (mg/dl), HDL-C (mg/dl), LDL-C (mg/dl), LDL/HDL ratio and TC/HDL-C ratio (Bastianelli et al., 2016). Since TC values <100mg/dl could not be measured by the instrument, measurements that result <100 mg/dl were interpreted as 100 mg/dl for the calculations in Friedewald's formula to estimate LDL-C values.

2.2.3 Body composition

Body composition was accessed with a multi-frequency bioelectrical impedance instrument called InBody 770. Measurements include values of the participants body weight, FFM, FM, BF% and body mass index (BMI). Upon arrival they were instructed to empty their bladders prior to the test and asked to perform the test in underwear for optimal results (or light clothes, as long as they felt comfortable). Accessories were removed prior to the test.

The subject stood on the platform of the instrument barefoot on the metal part to connect the electrodes. Then they grasped the handles and put their thumbs and fingers on the metal part. The participants stood still for approximately 1 minute with their elbows extended, and shoulders slightly abducted in approximately a 30-degree angle to make sure the arms did not touch the thorax (Antonio et al., 2019).

2.2.4 Waist circumference and height

During the measurement of waist circumference, the participants were instructed to stand upright, close with their feet together, body weight evenly distributed, arms at the side and wearing light or little clothing. The participants were then instructed to stand as relaxed as possible, as the instructor placed the measuring tape in the middle between the lower part of the last palpable rib and the top of the iliac crest. The measurements were executed at the end of a normal expiration and were performed twice. If the results differed more than 1cm the measurements were repeated, otherwise the average of the two measurements were calculated (WHO, 2008). The participants' height was measured two times, where the average height was noted.

2.3 Statistical analyses

All statistical analyzes were performed using the IBM SPSS Statistics 28 tool and the level of statistical significance (p) was set at 0.05 for all tests. Testing for normality was done with the Shapiro-Wilks test.

To determine associations between blood lipids, METS and time sedentary, we utilized Pearson’s correlation analysis for parametric data and Spearman's rank correlation analysis for non-parametric data to determine correlations coefficients (r). The correlations were deemed poor if r = ±0.01-0.2, fair if 0.3-0.5, moderate if r = ±0.6-0.7 and very strong if r = ± 0.8-0.9 (Akoglu, 2018). Coefficients of determination (R^2) were used to determine the strength of the correlations between variables.

To adjust our associations for different variables, we used ANOVA. METS and time sedentary were set as independent values and blood lipid variables, BF%, visceral fat area, waist circumference, BMI, skeletal muscle mass and weight circumference were tested separately as dependent variables.

2.4 Ethical and social considerations

This study involved humans and related data regarding young adults. The study followed the ethical principles of the declaration of Helsinki (WMA, 2022) which states that the health of our test persons must be our first consideration. All the participants were informed about the conditions to participate in the study before and have signed a written informed consent (see Appendix A). Participation was fully voluntary, and participants were able to cancel at any time without giving any further explanations. All collected personal data was treated by the students responsible for the study and their

supervisor, only. Personal data was coded and was presented at group level, and the code key was separated from the data so that unauthorized persons couldn't get access to it. All blood samples were thrown away immediately after the test and the data was saved until the work was approved. If the participants wanted to access their personal data, they could request it from the students in charge.

Possible risks involved physical pain, emotional discomfort, nausea and/ or anxiousness before, during and after the puncture to draw capillary blood test for lipid profile analysis, as well as psychological discomfort regarding the assessment of weight and body composition, which was prevented by being measuring the subjects in a secluded area. More long-term effects may include bruising or tenderness in the fingertips. To minimize these risks, we used necessary and proper hygiene, and made the test as comfortable as possible for our subjects. All disconcerns were taken seriously and respectfully.

Although there were some possible risks and discomforts coming with the test, the benefits of the study were considered worth it. Globally, there is a rise in cardiovascular diseases and was in 2019 responsible for 32% of all global deaths (WHO, 2021) and approximately 52% of deaths in Sweden were caused by CVD in 2021 (Socialstyrelsen, 2022). Since blood lipids, and physical activity are correlated with CVD it is of high importance to increase the knowledge regarding the subject. This study may provide insight into the health status of young men and women regarding blood lipids and physical activity. Hopefully, the results of this study will contribute to more extensive studies in the future and possibly increase the amount of people in Sweden that fulfill the physical activity recommendations (Piercy et al., 2018), to minimize diseases in the population. The goal with this study is also in line with the third goal, good health and wellbeing of Agenda 2030's sustainability goals, which aims to reduce the number of deaths caused by noncommunicable disease like CVD (UN, 2024).

3. Results

3.1 Group analysis

Fifteen (n=15) students with a mean age of 22.5 ± 1.4 fulfilled the inclusion criteria and participated in the study, 10 women and 5 men (table 1). The group had varying levels of activity, but the majority of subjects were active with a median of 3240 METS (IQR = 2060, 4540).

Table 1. Descriptive statistics of subjects included in this study (n=15).

	Mean (SD)
Age (yrs)	22.5 ± 1.4
Height (cm)	173.4 ± 11.5
Waist circumference (cm)	78.1 ± 9.4
Visceral fat area (cm^2)	75 ± 29.7
Percent body fat (%)	23.9 ± 6.8
BMI (kg/m^2)	23.9 ± 2.8
Skeletal muscle mass (kg)	31 ± 8.1
Weight (kg)	72.3 ±14.7
TC (mg/dl)	136.5 ± 32.7
HDL-C (mg/dl)	58.1 ± 13.6
TG (mg/dl)	102.7 ± 50.5
LDL-C (mg/dl)	57.9 ± 23.2
TC/HDL	2.4 ± 0.4
LDL/HDL	1 ± 0.4
METS (min/week)	3989.9 ± 3075.5
Sedentary time (h)	8.3 ± 2.2

SD = Standard deviation, BMI = Body Mass Index; TC = Total Cholesterol; HDL-C = High-density Lipoprotein Cholesterol; TG = Triglycerides; LDL-C = Low-density Lipoprotein Cholesterol; METS = Metabolic Equivalent of Task.

3.2 Correlation between physical activity level and blood lipids

In order to determine associations between physical activity levels and blood lipid levels we analyzed the correlation between METS and TC, HDL-C, TG, LDL-C and TC/HDL-C ratio separately. This resulted in a poor correlation ($r<0.2$) for all variables (table 2).

Table 2. Spearman's rank correlation between METS and TC, HDL-C, TG, LDL-C and TC/HDL-C (n=15).

		TC	HDL-C	TG	LDL-C	TC/HDL-C
METS	Spearman's rank Correlation coefficient (r)	0.143	0.145	-0.156	0.184	-0.015
	Sig. (2-tailed)	0.610	0.607	0.579	0.511	0.959

TC = Total Cholesterol; HDL-C = High-density Lipoprotein Cholesterol; TG = Triglycerides; LDL-C = Low-density Lipoprotein Cholesterol; METS = Metabolic Equivalent of Task.

3.3 Correlation between sedentary time and blood lipids

Associations between sedentary time and blood lipid levels were analyzed and resulted in a fair ($r=0.354$) correlation between sedentary time and TC/HDL-C ratio and a moderate correlation ($r=0.623$) between sedentary time and TG (table 3). The R^2 value indicates that 12.5% of the TC/HDL-C ratio (figure 5) and 35.8% of the TG values (figure 6) could be explained by factors involved in sedentary time. No difference in results when excluding the outlier from correlation of sedentary time and TG (figure 6).

Table 3. Pearson's correlation between sedentary time and TC, HDL-C, TG, LDL-C and TC/HDL-C (n=15).

		TC	HDL-C	TG*	LDL-C	TC/HDL-C
Sedentary time	Pearson's correlation coefficient (r)	0.196	-0.073	0.623	0.0063	0.354
	Sig. (2-tailed)	0.483	0.797	0.013	0.822	0.196

*= Spearman's rank correlation

TC = Total Cholesterol; HDL-C = High-density Lipoprotein Cholesterol; TG = Triglycerides; LDL-C = Low-density Lipoprotein Cholesterol; METS = Metabolic Equivalent of Task.

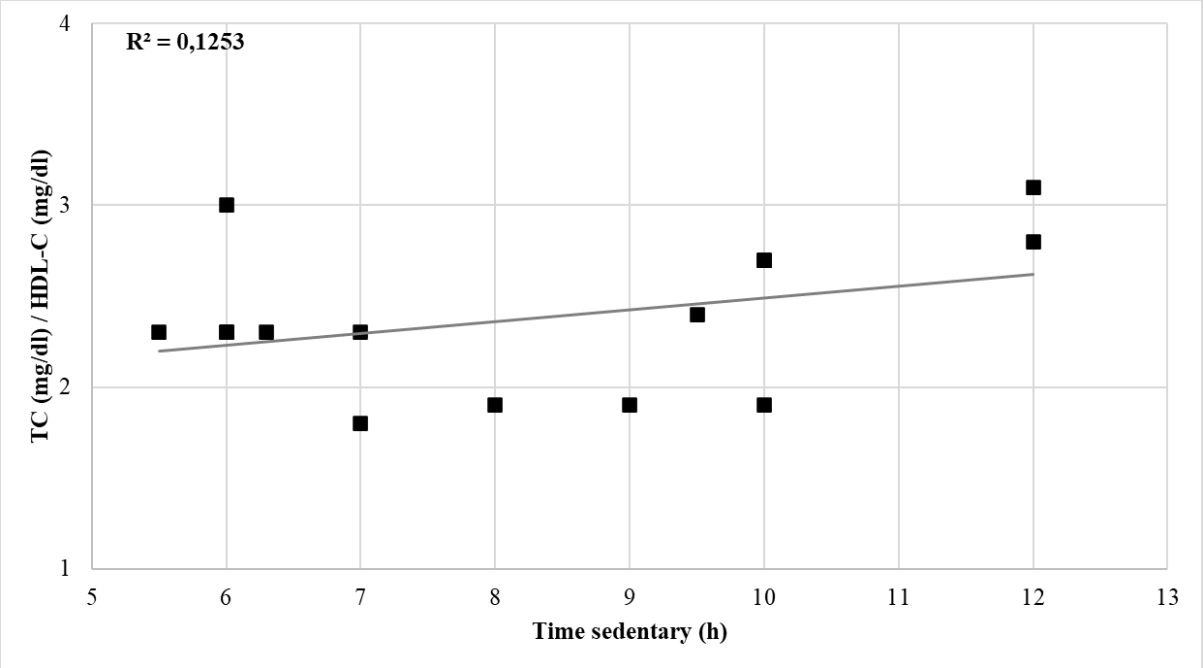

Figure 5. Scatter plot for sedentary time compared with TC/HDL-C. TC = Total cholesterol, HDL-C = High-density Lipoprotein Cholesterol (n=15).

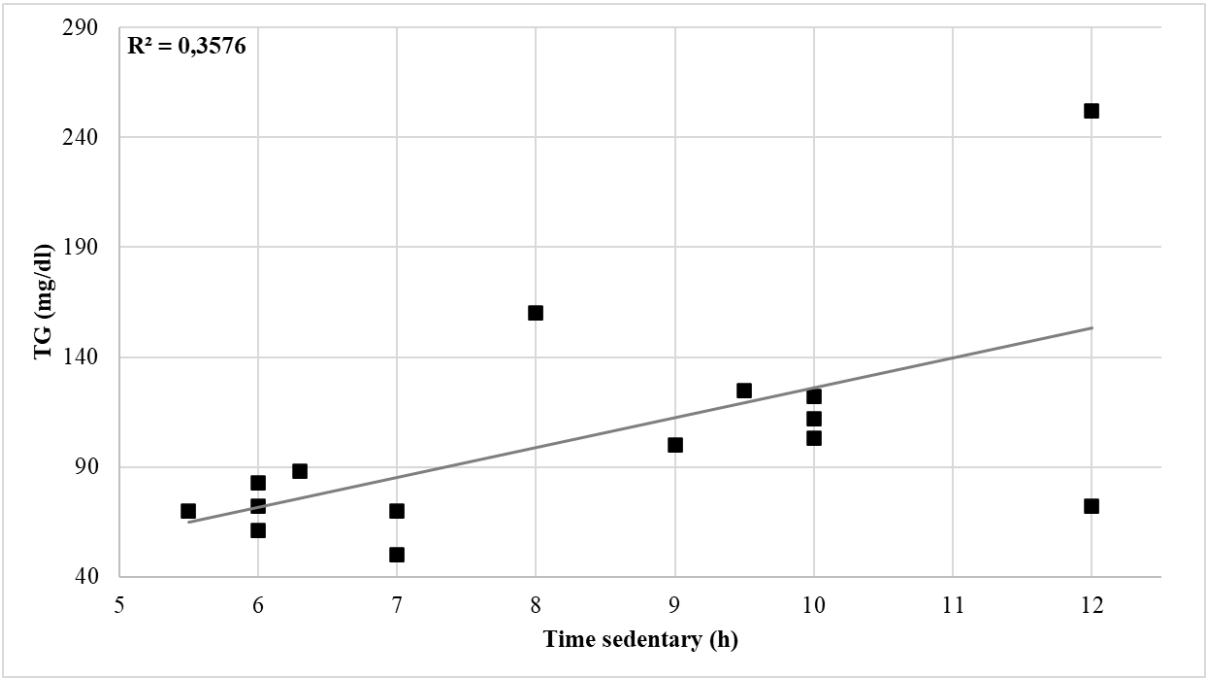

Figure 6. Scatter plot for sedentary time compared with TG. TG= Triglycerides (n=15).

3.4 Regression analysis

Associations between TG and time sedentary were analyzed with linear regressions and resulted in an unstandardized β-value of 13.8 (CI=2.2-25.5) p= 0.024, which entails that for every unit of time sedentary the TG value is expected to increase by 13.8 mg/dl. This remained true when adjusting for age and gender. When TG and METs were checked for association, there was not a statistical significance. TC/HDL-C ratio and time sedentary showed no statistical linearity.

4. Discussion

4.1 Primary findings

The results show that there was no statistical correlation between self estimated physical activity and blood lipid levels in this population, even when adjusting for age and gender. A moderate correlation between TG and time sedentary was observed, and a fair correlation between the TC/HDL-C ratio and time sedentary. There was an association between TG and time sedentary, and this remained true when adjusting for age, gender, visceral fat area, skeletal muscle mass and BMI. TC/HDL-C and time sedentary did not show statistical significance when tested for association.

4.2 Results discussion

The average MET value of 3989.9 indicates that this population is physically active although previous studies have shown that self reported physical activity tends to be

overestimated. Previous studies have shown that physical activity has a lowering effect on TC and LDL-C levels (Mann et al., 2014). The weak correlations between METS and blood lipids contradict the study by Mann et al (2014) which may be due to the high physical activity level and homogeneity of the group. According to Kyu et al (2016) most health gains and reduced risk for several diseases occur when individuals reach a physical activity level at 3000-4000 MET minutes/ week. Since our group reached an average MET value of 3989.9 it could be an explanation why they all are in relatively good health conditions regarding their blood lipid levels and we got a weak correlation. According to the Public Health Agency of Sweden (2024) 56.5% of Swedish men and women aged 16-29 are not active for more than 1200 MET minutes/week. The participants in the current study were not fully representative regarding the physical activity level of an average Swedish individual in this age range. It would have been interesting with a wider spectrum of activity levels.

The result in the present study shows a fair correlation between sedentary time and TC/HDL-C and a moderate correlation between sedentary time and TG. Our findings show that time sedentary may be an even more important variable for TG levels than time spent being physically active as in line with prior study by Celis-Morales et al (2012). As the values of sedentary time and TG levels increased linearly, meaning that it is more important to reduce sedentary behavior than to be physically active. Only one participant did have breakfast 2 hours before the test occasion, which was the individual with elevated TG levels; the rest of the group stayed fasted overnight. However, even when excluding the outlier from the analyzes no difference occurred on the results.

4.3 Methods discussion

One limitation in the current study that could have an impact on the results is that the data revolving the subjects diet was not included. As mentioned in the background section, diet has an impact on cholesterol- and lipid levels. Studies that compared vegetarian diets with omnivores shows that vegetarians and vegans tend to have better cardiovascular markers, and dietary interventions shows that vegans and vegetarians improve their lipid profile by lowering TC, TG and LDL-C (Lynch et al., 2018).

Self-reported physical activity questionnaires have both advantages and disadvantages. They are relatively quick, simple to perform, low-cost and can reach out to a large population. However, studies have shown that people tend to overestimate their physical activity level when doing the questions regarding moderate-to-vigorous physical activity in GPAQ in combination with an underestimation of their sedentary behavior (Keating et al., 2019). In the current study, most of the participants had never filled in a self-

reported physical activity questionnaire before which could have an impact on the results.

CardioChek Plus was an alternative way to measure the participants blood lipids to avoid hospital visits, extra costs and be able to do all tests in one location during one occasion. Strengths with the instrument is that controls were made for every newly opened batch, where the batches used passed the control. The instrument also has an acceptable accuracy compared to Roche Cobas analyser and is suitable for POC tests (Bastianelli et al., 2017). Limitations were that the measuring stick of the instrument required more blood than expected and was very sensitive which probably should have been taken into consideration before puncturing the finger. The instrument showed "error" when small samples were added, which led to several attempts for some individuals, but results for all participants were achieved and had no negative impact on the results. A more sharp and longer lancet to cut deeper would have been preferred. Also prepare the participant by warming up the hand to achieve a better blood flow. Furthermore, the instrument was only able to analyze TC levels >100 mg/dl which could have influenced the results. However, only two participants did not surpass the 100 mg/dl threshold and had to be set as 100 mg/dl for the statistical analysis.

Since CVDs are on a rise and currently is the leading cause of death in Sweden, ways to prevent and be more aware of individuals' health at an earlier stage needs to be found. Using a portable testing instrument like CardioChek Plus could be a feasible way to perform continuous controls of an individual's lipid profile, to get a good insight before damage has already occurred. Some individuals may not like the hospital environment and prefer capillary tests over venous samples. Could be a good complement for companies to utilize, to screen employee's health, which can catch individuals outside of the reference values. In the long run, this can help the burden on the healthcare system and reduce costs of hospital visits and treatments if individuals at an earlier stage know if they are at risk.

The use of Inbody 770 was an easy to use method to get descriptive data for our subjects and was deemed valid enough for this purpose. For a more accurate estimation of FFM, FM and BF%, DXA would be the preferable instrument, however we did not have access to it. Since Inbody 770 overestimates FFM and underestimates FM and BF% it could have a minor impact on our results (Antonio et al., 2019). But since these are not primary variables, a more expensive alternative could not be motivated for this study.

4.4 Study limitations

The primary limitation of this study was the small sample size, only consisting of 15 participants, a larger sample size could enable more statistical tests to utilize and check for sex differences. Another limitation is that the participants were generally not representative of the average Swedish individual in the aspect of physical activity. This may be because the Inbody 770 test attracts a population that invests in their personal health and are physically active.

5. Conclusion

In the studied population time spent sedentary is a more important factor to reduce TG levels than being physically active. Suggesting that individuals who want to decrease their risk of developing CVDs should not only be satisfied by reaching the recommended levels of physical activity each week, but also focus on reducing their time spent sedentary throughout the day.

5.1 Future studies

Further studies could utilize the current method with a larger group of participants to achieve deeper statistical analysis regarding the subject. Since the CardioChek plus analyzer is good for POC testing it could be possible to collect blood lipid profile data in a broader sense. A larger set of participants could also lead to a wider range of physical activity levels between the individuals which is of interest since our study mainly found highly physically active people. Future studies could use an objective measurement of physical activity level to further develop the method section, in the form of an accelerometer/ pedometer to reduce the impact of over- or underestimation. Future studies should also take diet into account if possible since it has a long-term effect on blood lipid levels. Sex differences are another interesting factor to take into consideration.

6. References

Abrahamsson, L., Andersson, A., Nilsson, G. (2013). *Näringslära för högskolan*. Liber

Akoglu H. (2018). User's guide to correlation coefficients. *Turkish journal of emergency medicine*, *18*(3), 91–93. https://doi.org/10.1016/j.tjem.2018.08.001

Alves-Bezerra, M., & Cohen, D. E. (2017). Triglyceride Metabolism in the Liver. *Comprehensive Physiology*, *8*(1), 1–8. https://doi.org/10.1002/cphy.c170012

Alonso-Herranz, L., Albarrán-Juárez, J., & Bentzon, J. F. (2023). Mechanisms of fibrous cap formation in atherosclerosis. *Frontiers in cardiovascular medicine*, *10*, 1254114. https://doi.org/10.3389/fcvm.2023.1254114

Antonio, J., Kenyon, M., Ellerbroek, A., Carson, C., Burgess, V., Tyler-Palmer, D., Mike, J., Roberts, J., Angeli, G., & Peacock, C. (2019). Comparison of Dual-Energy X-ray Absorptiometry (DXA) Versus a Multi-Frequency Bioelectrical Impedance (InBody 770) Device for Body Composition Assessment after a 4-Week Hypoenergetic Diet. *Journal of functional morphology and kinesiology*, *4*(2), 23. https://doi.org/10.3390/jfmk4020023

Ashorobi, D., Ameer, M. A., & Fernandez, R. (2023). Thrombosis. In *StatPearls.* StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK538430/

Bailey A., Mohiuddin S.S. (2022). Biochemistry, High Density Lipoprotein. *Treasure Island (FL): StatPearls Publishing;* https://www.ncbi.nlm.nih.gov/books/NBK549802/

Bastianelli, K., Ledin, S., & Chen, J. (2017). Comparing the Accuracy of 2 Point-of-Care Lipid Testing Devices. *Journal of pharmacy practice*, *30*(5), 490–497. https://doi.org/10.1177/0897190016651546

Bouhairie, V. E., & Goldberg, A. C. (2015). Familial hypercholesterolemia. *Cardiology clinics*, *33*(2), 169–179. https://doi.org/10.1016/j.ccl.2015.01.001

Celis-Morales, C. A., Perez-Bravo, F., Ibañez, L., Salas, C., Bailey, M. E., & Gill, J. M. (2012). Objective vs. self-reported physical activity and sedentary time: effects of measurement method on relationships with risk biomarkers. PloS one, 7(5), e36345. https://doi.org/10.1371/journal.pone.0036345

Chemistry LibreTexts. (2024, 27 May). *Lipids and Triglycerides.* https://chem.libretexts.org/Courses/University_of_Kentucky/CHE_103%3A_Chemistry_for_Allied_Health_%28Soult%29/14%3A_Biological_Molecules/14.02%3A_Lipids_and_Triglycerides

Cleveland Clinic. (2022, 22 May). *Lipoproteins.* https://my.clevelandclinic.org/health/articles/23229-lipoprotein

Elliott W. J. (2007). Systemic hypertension. *Current problems in cardiology*, *32*(4), 201–259. https://doi.org/10.1016/j.cpcardiol.2007.01.002

Fagard R. H. (2001). Exercise characteristics and the blood pressure response to dynamic physical training. *Medicine and science in sports and exercise*, *33*(6 Suppl), S484–S494. https://doi.org/10.1097/00005768-200106001-00018

Falk E. (2006). Pathogenesis of atherosclerosis. *Journal of the American College of Cardiology*, *47*(8 Suppl), C7–C12. https://doi.org/10.1016/j.jacc.2005.09.068

Fang, H., Berg, E., Cheng, X., & Shen, W. (2018). How to best assess abdominal obesity. *Current opinion in clinical nutrition and metabolic care*, *21*(5), 360–365. https://doi.org/10.1097/MCO.0000000000000485

Feingold, K. R. (2024). Introduction to Lipids and Lipoproteins. In K. R. Feingold (Eds.) et. al., *Endotext*. MDText.com, Inc. https://www.ncbi.nlm.nih.gov/books/NBK305896/

Feingold, K. R. (2021). The Effect of Diet on Cardiovascular Disease and Lipid and Lipoprotein Levels. In K. R. Feingold (Eds.) et. al., *Endotext.* MDText.com, Inc. https://www.ncbi.nlm.nih.gov/books/NBK570127/

Franczyk, B., Gluba-Brzózka, A., Ciałkowska-Rysz, A., Ławiński, J., & Rysz, J. (2023). The Impact of Aerobic Exercise on HDL-C Quantity and Quality: A Narrative Review. *International journal of molecular sciences*, *24*(5), 4653. https://doi.org/10.3390/ijms24054653

Gallagher, D., Heymsfield, S. B., Heo, M., Jebb, S. A., Murgatroyd, P. R., & Sakamoto, Y. (2000). Healthy percentage body fat ranges: an approach for developing guidelines based on body mass index. *The American journal of clinical nutrition*, *72*(3), 694–701. https://doi.org/10.1093/ajcn/72.3.694

Gimbrone, M. A., Jr, & García-Cardeña, G. (2016). Endothelial Cell Dysfunction and the Pathobiology of Atherosclerosis. *Circulation research*, *118*(4), 620–636. https://doi.org/10.1161/CIRCRESAHA.115.3063

Grześk, G., Dorota, B., Wołowiec, Ł., Wołowiec, A., Osiak, J., Kozakiewicz, M., & Banach, J. (2022). Safety of PCSK9 inhibitors. *Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie*, *156*, 113957. https://doi.org/10.1016/j.biopha.2022.113957

Huang P. L. (2009). A comprehensive definition for metabolic syndrome. *Disease models & mechanisms*, *2*(5-6), 231–237. https://doi.org/10.1242/dmm.001180

Jeong, S. W., Kim, S. H., Kang, S. H., Kim, H. J., Yoon, C. H., Youn, T. J., & Chae, I. H. (2019). Mortality reduction with physical activity in patients with and without cardiovascular disease. *European heart journal*, *40*(43), 3547–3555. https://doi.org/10.1093/eurheartj/ehz564

Keating, X. D., Zhou, K., Liu, X., Hodges, M., Liu, J., Guan, J., Phelps, A., & Castro-Piñero, J. (2019). Reliability and Concurrent Validity of Global Physical Activity Questionnaire (GPAQ): A Systematic Review. *International journal of environmental research and public health*, *16*(21), 4128. https://doi.org/10.3390/ijerph16214128

Keirns, B. H., Sciarrillo, C. M., Koemel, N. A., & Emerson, S. R. (2021). Fasting, non-fasting and postprandial triglycerides for screening cardiometabolic risk. Journal of nutritional science, 10, e75. https://doi.org/10.1017/jns.2021.73

Kopčeková, J., Holovičová, M., Gažarová, M., Mrázová, J., Habánová, M., Mečiarová,

L., & Bronkowska, M. (2020). Association between Selected Dietary Habits and Lipid Profiles of Patients with Cardiovascular Disease. *International journal of environmental research and public health*, *17*(20), 7605. https://doi.org/10.3390/ijerph17207605

Krishnaveni, P., & Gowda, V. M. (2015). Assessing the Validity of Friedewald's Formula and Anandraja's Formula For Serum LDL-Cholesterol Calculation. *Journal of clinical and diagnostic research : JCDR*, *9*(12), BC01–BC4. https://doi.org/10.7860/JCDR/2015/16850.6870

Kyu, H. H., Bachman, V. F., Alexander, L. T., Mumford, J. E., Afshin, A., Estep, K., Veerman, J. L., Delwiche, K., Iannarone, M. L., Moyer, M. L., Cercy, K., Vos, T., Murray, C. J., & Forouzanfar, M. H. (2016). Physical activity and risk of breast cancer, colon cancer, diabetes, ischemic heart disease, and ischemic stroke events: systematic review and dose-response meta-analysis for the Global Burden of Disease Study 2013. *BMJ (Clinical research ed.)*, *354*, i3857. https://doi.org/10.1136/bmj.i3857

Linton, M. F., Yancey, P. G., Davies, S. S., Jerome, W. G., Linton, E. F., Song, W. L., Doran, A. C., & Vickers, K. C. (2019). The Role of Lipids and Lipoproteins in Atherosclerosis. In K. R. Feingold (Eds.) et. al., *Endotext*. MDText.com, Inc. https://www.ncbi.nlm.nih.gov/books/NBK343489/

Lynch, H., Johnston, C., & Wharton, C. (2018). Plant-Based Diets: Considerations for Environmental Impact, Protein Quality, and Exercise Performance. *Nutrients*, *10*(12), 1841. https://doi.org/10.3390/nu10121841

Madan, K., & Sawhney, J. P. S. (2024). Exercise and lipids. Indian heart journal, 76 Suppl 1(Suppl 1), S73–S74. https://doi.org/10.1016/j.ihj.2023.11.270

Mann, S., Beedie, C., & Jimenez, A. (2014). Differential effects of aerobic exercise, resistance training and combined exercise modalities on cholesterol and the lipid profile: review, synthesis and recommendations. *Sports medicine (Auckland, N.Z.)*, *44*(2), 211–221. https://doi.org/10.1007/s40279-013-0110-5

McLester, C. N., Nickerson, B. S., Kliszczewicz, B. M., & McLester, J. R. (2020). Reliability and Agreement of Various InBody Body Composition Analyzers as Compared to Dual-Energy X-Ray Absorptiometry in Healthy Men and Women. *Journal*

of clinical densitometry : the official journal of the International Society for Clinical Densitometry, *23*(3), 443–450. https://doi.org/10.1016/j.jocd.2018.10.008

MedlinePlus. (2020, 2 October). *Cholesterol Levels: What You Need to Know*. https://medlineplus.gov/cholesterollevelswhatyouneedtoknow.html

Mukaka M. M. (2012). Statistics corner: A guide to appropriate use of correlation coefficient in medical research. *Malawi medical journal : the journal of Medical Association of Malawi*, *24*(3), 69–71.

Mushenkova, N. V., Summerhill, V. I., Zhang, D., Romanenko, E. B., Grechko, A. V., & Orekhov, A. N. (2020). Current Advances in the Diagnostic Imaging of Atherosclerosis: Insights into the Pathophysiology of Vulnerable Plaque. *International journal of molecular sciences*, *21*(8), 2992. https://doi.org/10.3390/ijms21082992

Nordestgaard, B. G., Langsted, A., Mora, S., Kolovou, G., Baum, H., Bruckert, E., Watts, G. F., Sypniewska, G., Wiklund, O., Borén, J., Chapman, M. J., Cobbaert, C., Descamps, O. S., von Eckardstein, A., Kamstrup, P. R., Pulkki, K., Kronenberg, F., Remaley, A. T., Rifai, N., Ros, E., … European Atherosclerosis Society (EAS) and the European Federation of Clinical Chemistry and Laboratory Medicine (EFLM) joint consensus initiative (2016). Fasting is not routinely required for determination of a lipid profile: clinical and laboratory implications including flagging at desirable concentration cut-points-a joint consensus statement from the European Atherosclerosis Society and European Federation of Clinical Chemistry and Laboratory Medicine. *European heart journal*, *37*(25), 1944–1958. https://doi.org/10.1093/eurheartj/ehw152

Nystoriak, M. A., & Bhatnagar, A. (2018). Cardiovascular Effects and Benefits of Exercise. *Frontiers in cardiovascular medicine*, *5*, 135. https://doi.org/10.3389/fcvm.2018.00135

Saklayen M. G. (2018). The Global Epidemic of the Metabolic Syndrome. *Current hypertension reports*, *20*(2), 12. https://doi.org/10.1007/s11906-018-0812-z

Sigurdsson, A. F. (2017, 30 January). *VLDL – The Role of Triglyceride-Rich Lipoproteins and Remnant Cholesterol.* https://www.docsopinion.com/vldl-triglyceride-remnant-cholesterol/

Singh, P., Covassin, N., Marlatt, K., Gadde, K. M., & Heymsfield, S. B. (2021). Obesity, Body Composition, and Sex Hormones: Implications for Cardiovascular Risk. *Comprehensive Physiology*, *12*(1), 2949–2993. https://doi.org/10.1002/cphy.c210014

Sirtori, C.R. (2014). The pharmacology of statins. *Pharmacological Research, 88*, 3-11. https://doi.org/10.1016/j.phrs.2014.03.002

Socialstyrelsen. (2022). *Statistik om dödsorsaker år 2021*. https://www.socialstyrelsen.se/globalassets/sharepoint-dokument/artikelkatalog/statistik/2022-6-8020.pdf

Piercy, K. L., Troiano, R. P., Ballard, R. M., Carlson, S. A., Fulton, J. E., Galuska, D. A., George, S. M., & Olson, R. D. (2018). The Physical Activity Guidelines for Americans. JAMA, 320(19), 2020–2028. https://doi.org/10.1001/jama.2018.14854

Pts diagnostics. (2024). *CardioChek Plus.* https://ptsdiagnostics.com/cardiochek-plus-analyzer/

Public Health Agency of Sweden. (2024, 24 April). *Fysisk aktivitet (självrapporterat) efter ålder, kön och år. Andel (procent).* http://fohm-app.folkhalsomyndigheten.se/Folkhalsodata/sq/24111611-c04d-4820-8702-bcb2e624de98

United Nations. (2024, 24 April). *Goal 3: Ensure healthy lives and promote well-being for all at all ages.* https://www.un.org/sustainabledevelopment/health/

Vacek, T. P., Rehman, S., Neamtu, D., Yu, S., Givimani, S., & Tyagi, S. C. (2015). Matrix metalloproteinases in atherosclerosis: role of nitric oxide, hydrogen sulfide, homocysteine, and polymorphisms. *Vascular health and risk management*, *11*, 173–183. https://doi.org/10.2147/VHRM.S68415

World health organization. (2008, 8-11 December). *Waist Circumference and Waist-Hip Ratio Report of a WHO Expert Consultation* https://www.who.int/publications/i/item/9789241501491

World health organization. (2021). *Cardiovascular diseases (CVDs)*. https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)

World health organization. (2023, 16 March). *Hypertension.* https://www.who.int/news-room/fact-sheets/detail/hypertension

World health organization. (2024, 1 April). *Global Physical Activity Questionnaire (GPAQ) Analysis Guide.* https://www.who.int/docs/default-source/ncds/ncd-surveillance/gpaq-analysis-guide.pdf

World medical association. (2022, 6th September). *WMA DECLARATION OF HELSINKI – ETHICAL PRINCIPLES FOR MEDICAL RESEARCH INVOLVING HUMAN SUBJECTS.* https://www.wma.net/policies-post/wma-declaration-of-helsinki-ethical-principles-for-medical-research-involving-human-subjects/

7. Appendices

Appendix A

Informed consent

Information till dig som ska delta i en undersökning kring fysisk aktivitet och blodfetter som genomförs av studenter under utbildning

Information till forskningspersoner

Vi vill fråga dig om du vill delta i studentprojektet Jämförelse av blodfetter hos unga vuxna i Sverige baserat på fysisk aktivitetsnivå. I det här dokumentet får du information om projektet och om vad det innebär att delta.

Vad är det för ett projekt och varför vill ni att jag ska delta

Denna information får du för att du visat intresse för denna studie.

Hjärt- och kärlsjukdomar är ett stort samhällsproblem i dagsläget som bland annat kan kopplas till att stillasittande idag kan bidra till ökad sjuklighet i framtiden. Nivåer av blodfetter kan användas för att bedöma risken att utveckla hjärt-och kärlsjukdomar och tidigare forskning visar att fysisk aktivitet är en effektiv behandlingsmetod för att

förebygga utveckling av hjärt-och kärlsjukdomar. I denna studie kommer vi att undersöka hur blodfettnivåer ser ut hos svenska män och kvinnor som är olika fysiskt aktiva i åldrarna 18-25, detta för att se hur hälsostatusen ser ut i dagsläget. Du har blivit tillfrågad att delta för att du är född mellan 1999-2006 och är därav relevant för vår studie, samt visat intresse.

Forskningshuvudman för projektet är Högskolan i Halmstad. Med forskningshuvudman menas den organisation som är ansvarig för projektet. Ansökan har godkänts av handledare och ansvariga för utbildningen.

Hur går projektet till?

För att delta i denna studie behöver du ge samtycke för ditt deltagande, via informerat samtycke får du information kring innebörd, eventuella risker, fördelar, frivillighet att delta och rätt att avbryta närsomhelst. Vi ber dig att avstå från att delta om du har någon hjärt-och kärlsjukdom, familjär hyperkolesterolemi regelbundet tar medicin för någon sjukdom som påverkar kolesterol eller röker regelbundet.

Om du vill delta kommer du bli tilldelad en tid på rörelselabbet för testerna som passar dig, inför testet ber vi dig att komma med ombyte till lätt klädsel då det krävs för ett av testerna. Du som deltagare behöver undvika intag av mat och dryck två timmar innan testet, samt hård träning. Vid ankomst till testtillfället kommer du att vila 10 minuter innan mätningen, där du under vilan kommer fylla i ett frågeformulär som kallas GPAQ där man fyller i vanor kopplade till fysisk aktivitet. Mätningen involverar ett stick i fingret där ungefär en droppe blod kommer att absorberas av en mätsticka och analyseras av mätinstrumentet CardioChek PA. Mätstickan med det kapillära blodet slängs direkt efter analys. Därefter kommer vi mäta din längd, midjemått och uppskatta din kroppssammansättning med ett instrument som kallas InBody 770. För dig som vill är det möjligt att ta del av dina värden på plats. Totala tiden för testtillfället kräver runt 20-30 minuter.

Möjliga följder och risker med att delta i projektet

Möjliga risker kan innefatta fysisk smärta, känslomässigt obehag, illamående och/eller ångest före, under och efter punkteringen av hud på fingret för att ta ett blodprov. Kan upplevas jobbigt att väga sig i en InBody 770 mätning och mätning av midjemått då du som testperson behöver ha lätt klädsel, men detta görs enskilt på en avskild plats för att minimera obehaget. Mer långsiktiga effekter kan inkludera blåmärken eller ömhet i det punkterade fingret. För att minimera dessa risker kommer vi att använda nödvändiga hygienrutiner och göra testet så bekvämt som möjligt för dig som deltar. Mängden blod

är så liten att den inte förväntas påverka dig. All visad oro och osäkerhet kommer att tas på allvar och med respekt. Om biverkningar uppstår efter test kontaktas studieledare, eller 1177. Utöver risker som kan komma med testet kan du som deltagare få information angående din hälsa avseende blodfettsnivåer, kroppssammansättning och insikt om hur fysiskt aktiv du är, helt utan kostnad.

Vad händer med mina uppgifter?

Projektet kommer att samla in information om ålder, längd, midjemått och kön, svar angående fysisk aktivitet, kroppssammansättning och blodfettsnivåer. Uppgifterna kommer att användas för att se samband mellan fysisk aktivitet och blodfetter. Resultaten redovisas på gruppnivå och ingen kommer att kunna se vem som fått vilka värden. Dessutom kommer personuppgifterna ersättas med kod som kommer att förvaras på en extern hårddisk som enbart ansvariga studenter och handledare har tillgång till i syfte att förhindra obehöriga att ta del av dem. Kodnyckeln kommer att förvaras på [Högskolan i Halmstad]. Uppgifterna kommer att raderas så snart arbetet godkänts.

Ansvarig för dina personuppgifter är Högskolan i Halmstad. Enligt EU:s dataskyddsförordning har du rätt att kostnadsfritt få ta del av de uppgifter om dig som hanteras i projektet, och vid behov få eventuella fel rättade. Du kan också begära att uppgifter om dig raderas samt att behandlingen av dina personuppgifter begränsas. Rätten till radering och till begränsning av behandling av personuppgifter gäller dock inte när uppgifterna är nödvändiga för den aktuella studien. Om du vill ta del av uppgifterna ska du kontakta Martin Olsen, martol21@student.hh.se, 070-9892880 alternativt Pontus Johansson, ponjoh21@student.hh.se , 072-2026111. Dataskyddsombudet nås på dataskydd@hh.se. Om du är missnöjd med hur dina personuppgifter behandlas har du rätt att ge in klagomål till Integritetsskydd Myndigheten, som är tillsynsmyndighet.

Vad händer med mina prover?

Det blod som samlas in för analys av blodfetter slängs direkt efter antecknat svar. Insamling av uppgifter kommer att följa lagar kring dataskyddsförordningen (GDPR).

Hur får jag information om resultatet av projektet?

Ni som deltagare har möjlighet att ta del av sina resultat angående fysisk aktivitet, kroppssammansättning och blodfettnivåer på plats av testen men har även full möjlighet att avstå från sina resultat. Vid avvikande fynd, enligt vårdens referensnivåer för dessa blodvärden kan vidare vård rekommenderas av ansvariga för studien.

Arbetet kommer att publiceras på DiVA (digitala vetenskapliga arkivet) https://hh.diva-portal.org där man kan ta del av resultatet, alternativt kontakta ansvariga studenter.

Försäkring och ersättning

Ni som deltar är försäkrad via personskadeförsäkring av Halmstad Högskola. Ingen ersättning ges ut för att delta.

Deltagandet är frivilligt

Ditt deltagande är frivilligt och du kan när som helst välja att avbryta deltagandet. Om du väljer att inte delta eller vill avbryta ditt deltagande behöver du inte uppge varför, och det kommer inte påverka dig på något sätt.

Om du vill avbryta ditt deltagande ska du kontakta de ansvariga för projektet (se nedan).

Ansvariga för projektet

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Appendix B

Global physical activity questionnaire (GPAQ)

Physical Activity			
Next I am going to ask you about the time you spend doing different types of physical activity in a typical week. Please answer these questions even if you do not consider yourself to be a physically active person. Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting for food, seeking employment. *[Insert other examples if needed]*. In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate.			
Questions		**Response**	**Code**
Activity at work			
1	Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like *[carrying or lifting heavy loads, digging or construction work*] for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P 4*	P1
2	In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	Number of days └┘	P2
3	How much time do you spend doing vigorous-intensity activities at work on a typical day?	Hours : minutes └┘└┘ : └┘└┘ hrs mins	P3 (a-b)
4	Does your work involve moderate-intensity activity that causes small increases in breathing or heart	Yes 1 No 2 *If No, go to P 7*	P4

	rate such as brisk walking *[or carrying light loads*] for at least 10 minutes continuously?		
5	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days └─┘	P5
6	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours : minutes └─┴─┘ : └─┴─┘ hrs min s	P6 (a-b)

Travel to and from places

The next questions exclude the physical activities at work that you have already mentioned.

Now I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship.

7	Do you walk or use a bicycle (*pedal cycle*) for at least 10 minutes continuously to get to and from places?	Yes 1 No 2 *If No, go to P 10*	P7
8	In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places?	Number of days └─┘	P8
9	How much time do you spend walking or bicycling for travel on a typical day?	Hours : minutes └─┴─┘ : └─┴─┘ hrs mins	P9 (a-b)

Recreational activities

The next questions exclude the work and transport activities that you have already mentioned.

Now I would like to ask you about sports, fitness and recreational activities (leisure).

10	Do you do any vigorous-intensity sports, fitness or recreational (*leisure*) activities that cause large increases in breathing or heart rate like [*running or football,]* for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P 13*	P10
11	In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (*leisure*) activities?	Number of days ⌊__⌋	P11
12	How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋ hrs mins	P12 (a-b)

Continued on next page

GPAQ, Continued

Physical Activity (recreational activities) contd.			
Questions		**Response**	**Code**
13	Do you do any moderate-intensity sports, fitness or recreational *(leisure*) activities that causes a small increase in breathing or heart rate such as brisk walking,(*cycling, swimming, volleyball*)for at least 10 minutes continuously?	Yes 1 No 2 *If No, go to P16*	P13
14	In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (*leisure*) activities?	Number of days ⌊__⌋	P14
15	How much time do you spend doing moderate-intensity sports,	Hours : minutes ⌊__⌋⌊__⌋ : ⌊__⌋⌊__⌋	P15 (a-b)

	fitness or recreational (*leisure*) activities on a typical day?	hrs mins	

Sedentary behaviour

The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent [sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television], but do not include time spent sleeping.

16	How much time do you usually spend sitting or reclining on a typical day?	Hours : minutes └┘└┘ : └┘└┘ hrs min s	P16 (a-b)